Use of Calcitonin in Sickle Cell Bone Crisis

To the Editor:

Despite recent therapeutic advances, many patients with sickle cell disease suffer from recurrent episodes of painful crises. Pain management of these acute events remains a significant issue, and problems of narcotic tolerance and complications are often encountered. Described is the first reported use of calcitonin to alleviate the bone pain of sickle crisis.

A 24-year-old man with SS disease complicated by recurrent crises and arthropathy presented to the emergency room with mild dyspnea, cough productive of purulent sputum, and chest and mid-back pain. On examination, he was afebrile but uncomfortable, with palpation of the chest wall and light percussion of the lower thoracic spine reproducing his musculoskeletal symptoms. Chest and thoracic spine radiographs were unremarkable. A complete blood count showed a minimally elevated white blood cell count and a hemoglobin level of 9.9 g/dL, with 29% reticulocytes.

The patient was treated with an intravenous antibiotic and hydration and required subcutaneous (SC) morphine sulfate at an average interval of 2.5 hours. The morning after admission he remained bedridden because of persistent mid-back and chest wall pain. After a test dose, treatment with salmon calcitonin at 100 IU SC was begun every 12 hours. Within 24 hours, the patient’s morphine requirements decreased 50%, and he began to ambulate. The patient was discharged home the following day on intranasal salmon calcitonin at 200 IU/d to complete a 2-week course of therapy, after which he was asymptomatic. His reported narcotic use when first home was approximately one-fourth that of previous discharges for crisis. Nausea and vomiting developed briefly within 2 hours of his second and third doses of SC salmon calcitonin; no adverse effects were reported from the intranasal preparation.

The utility of calcitonin in the treatment of acute vertebral fracture and phantom limb pain has been established in double-blind studies. The rate of improvement in our patient’s symptoms is consistent with previous reports, perhaps due to calcitonin’s ability to rapidly increase cerebrospinal b-endorphin levels, an effect that may explain, at least in part, its analgesic properties.

The potential benefit of using calcitonin in the treatment of sickle crisis bone pain syndromes is significant and worthy of further study. Initial use of the intranasal preparation should be considered, given its better side-effect profile and possibly enhanced analgesic effect.

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REFERENCES

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